Kaohsiung Journal of Medical Sciences (2015) 31, 156–162

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.kjms-online.com

ORIGINAL ARTICLE

Cerebral venous etiology of intracranial hypertension and differentiation from idiopathic intracranial hypertension

Stefan Mircea Iencean [a,b,*], **Ion Poeata** [a,b], **Andrei Stefan Iencean** [b], **Alexandru Tascu** [c]

[a] *Neurosurgery Department, GrT Popa University of Medicine and Pharmacy Iasi, Romania*
[b] *Neurosurgery Department, Prof N. Oblu Emergency Clinical Hospital Iasi, Romania*
[c] *Neurosurgery Department, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania*

Received 3 January 2014; accepted 1 September 2014
Available online 19 January 2015

KEYWORDS
Intracranial pressure;
Vascular intracranial hypertension;
Venous sinus stenosis;
Venous sinus thrombosis

Abstract This study presents the characteristics that distinguish between idiopathic intracranial hypertension (ICH) and ICH caused by intracranial vascular damage. Twenty-one patients with ICH were included in this study. The analysis of the symptomatology correlated with the values of intracranial pressure, and the imaging findings revealed significant differences between these two types of ICH. ICH caused by intracranial venous vascular damage is named vascular ICH. Vascular ICH has a known etiology, such as cerebral vascular illness, and a relatively rapid increase in intracranial pressure of approximately 21 cmH_2O and imaging findings show characteristic images of thrombosis or stenosis of the intracranial venous system, while all brain images (computed tomography, magnetic resonance imaging, angio-magnetic resonance imaging) are normal in idiopathic ICH. The treatment of vascular ICH is etiologic, pathogenic, and symptomatic, but that of idiopathic ICH is only symptomatic.

Conflicts of interest: All authors declare no conflicts of interest.

* Corresponding author. 12 Strap Silvestru Street, CL 18/5, 700006 Iasi, Romania.
E-mail address: mirceasteffan@yahoo.com (S.M. Iencean).

http://dx.doi.org/10.1016/j.kjms.2014.12.007

Introduction

Intracranial hypertension (ICH) is a common and significant problem in neurology and neurosurgery. The symptomatology consisting of cephalea, vomiting, and psychical disorders may be significant for ICH and, together with the finding of papillary edema, comprises the pathognomonic signs for ICH. The diagnosis is established by paraclinical investigations (to detect a tumor, traumatic brain injury, obstructive hydrocephalus, meningitis, etc.), but some of the cases are included in the category of idiopathic ICH without an identifiable cause [1,2]. Many authors have indicated that intracranial venous sinus thrombosis and stenosis are the main etiological factors for idiopathic ICH [3,4]. Other authors consider that there are differences between idiopathic ICH and ICH caused by sinus thrombosis or stenosis [5—7]. We believe that venous sinus pathological damage is a vascular cause of ICH and that these cases must be included in a vascular etiology of ICH, different of idiopathic ICH. The purpose of this study was to identify the clinical differences, the imaging findings, and etiological differences between cases of idiopathic ICH and cases of ICH caused by intracranial vascular damage. This distinction is very important because the therapy for the two is different: pathogenic and possible etiologic in vascular ICH or only symptomatic in idiopathic ICH.

Methods

Twenty-one patients (14 women and 7 men) aged 18—61 years, none of whom had a history of cerebrovascular disease, were included in a prospective study during the past 7 years. Upon admission they presented the clinical symptomatology of ICH with the absence of neurological location signs. The main symptom was diffuse cephalea of a progressive nature over a period of several days to several months. The eye fundus examinations showed different aspects, from the incipient aspect of papillary contour blur to the manifestation of papillary edema with venous stasis. The imaging diagnosis involved craniocerebral computed tomography (CT) and/or craniocerebral magnetic resonance imaging (MRI, with and without contrast), which showed the absence of an expansive intracranial process, hydrocephalus and intracranial infection. Cerebral angioMRI venous phases showed whether there was cerebral vascular disease, such as cerebral venous thrombosis, venous sinus thrombosis or stenosis. In some cases for the diagnosis, angioCT or cerebral Seldinger angiography was performed.

After ruling out intracranial lesions that could produce brain edema, by CT or cerebral MRI exploration, the pressure of the cerebrospinal fluid (CSF) was determined by repeated lumbar puncture with manometry (3—4 times).

Therefore we did the lumbar puncture after the imaging study and we repeated them during the treatment and after 3—4 weeks of treatment. In all patients, the following tests were performed: complete blood count, chemistry panel, prothrombin time, activated partial thromboplastin time, the composition of the CSF with the CSF proteins and the cellularity. The patients were followed for 3—12 months after their diagnosis. All procedures complied with the relevant European guidelines [8]. All patients gave their written informed consent before the start of the study, and the hospital ethics committee approved the study protocol (Neurosurgery Clinic, GrT Popa University of Medicine and Pharmacy Iasi, Romania).

Results

The patients' characteristics in admission order and the findings are shown in Table 1 and they can be grouped into two categories.

Twelve patients (3 men and 9 women, age 18—51 years) were diagnosed with idiopathic ICH based on the clinical symptomatology and the paraclinical investigation results: normal CT, or only small ventricles (Fig. 1), normal MRI, and normal angioMRI including the venous phase. Additionally the CSF had a normal composition with normal or reduced CSF proteins and normal cellularity. The pressure of the CSF was 22—30 cmH_2O with a mean of 27 cmH_2O.

The treatment was individualized for each patient and consisted of the following: control of headache with analgesics (e.g., paracetamol and amitriptyline); acetazolamide, which reduces CSF production, with control of the blood electrolyte levels; a weight-reduction diet for all patients, especially those who were obese [1,5,9]. In two cases, only the drainage of CSF by lumbar puncture was sufficient to control the symptomatology, and in one patient progressive visual loss in one eye responded well to oral prednisone.

The treatment during hospitalization spanned 2— 4 weeks with clinical improvement, with the resolution of papilledema, with CSF pressure returning to values of 10—12 cmH_2O and normal brain MRI controls. There was no need for surgery to correct the visual function deficit. The patients were observed every 4 weeks after discharge, and follow-ups were conducted from 3 months to 1 year. There were no major complications and the patients appeared to have stabilized with normal visual function at the end of the follow-up period.

Nine patients (4 men and 5 women, age 23—61 years) were diagnosed with ICH caused by intracranial vascular damage: acute, subacute or chronic thrombosis of the superior sagittal sinus; transverse sinus stenosis; or acute brain venous thrombosis. The brain CT (native, contrast, angioCT) showed brain edema in five cases, and the empty Δ sign in one case showed thrombosis of the superior sagittal sinus (a triangular filling defect—clot—in the sagittal sinus; Fig. 3A). The MRI and angioMRI with venous phase showed characteristic images of thrombosis or partial thrombosis of the intracranial venous sinus in five cases, of stenosis of the transverse sinus in three patients, and of acute cerebral venous thrombosis in one patient (Figs. 4—6). The pressure of the CSF for each patient was 19—26 cm H_2O with mean 21.8 cmH_2O.

The treatment began immediately and mainly consisted of the following: anticoagulation therapy to stop blood clot formation in cerebral venous sinus thrombosis, which was initially low-molecular-weight heparin and then oral anticoagulants in one case of acute cerebral venous thrombosis and in two cases of women with ICH caused by intracranial vascular damage in the postnatal period or only oral

Table 1 Patients' characteristics and findings (in order of admission).

Case	Age	Sex	Headache before admission	CT/MRI	AngioMRI + venous phase	CSF	Eye fundus	ICP cmH_2O before tx	ICP cmH_2O after tx	Follow-up	Observation
1	21	F	2 mo	N	N	N	Papilledema	29	12	8 mo	Tetracycline
2	32	F	4 mo	N	N	N	Venous stasis	27	11	1 y	Obesity
3	23	F	7 d	Brain edema	Part thromb SSS	N	Papilledema	21	8	6 mo	Postnatal
4	19	M	6 wk	N	N	N	Papilledema	26	10	6 mo	Obesity
5	41	F	7 mo	N	N	N	Venous stasis	26	12	7 mo	Obesity
6	32	M	3 wk	Brain edema	RTS stenosis	N	Papilledema	19	9	6 mo	
7	26	F	2 wk	N	RTS stenosis	N	Papilledema	18–20	11	6 mo	
8	18	M	9 wk	N	N	N	Venous stasis	27	12	9 mo	Obesity
9	31	F	4 mo	Small vent	N	N	Papilledema	29	12	6 mo	Obesity
10	55	M	2 mo	N	Part thromb LTS	N	Papilledema	19	10	4 mo	Left mastoiditis
11	31	F	3 mo	N	N	N	Venous stasis	27	12	4 mo	Obesity
12	29	F	5 mo	N	N	N	Papilledema	22	7	4 mo	Obesity
13	37	M	10 wk	Brain edema	Calcific SSS	N	Papilledema	19	11	3 mo	
14	35	F	7 mo	N	N	N	Papilledema	29	11	6 mo	
15	32	F	10 d	Δ sign	thromb SSS	N	Papilledema	24	11	1 y	Postnatal
16	32	F	3 mo	N	N	N	Papilledema	26	12	3 mo	Obesity
17	38	F	2 wk	Brain edema	Part thromb SSS	N	Venous stasis	25	11	1 y	Contraceptives
18	53	M	5 d	Brain edema	ACV thromb	N	Venous stasis	26	10	9 mo	Seizures
19	47	F	5 mo	N	N	N	Papilledema	30	9	6 mo	
20	42	F	2 mo	N	LTS stenosis	N	N	25	11	7 mo	
21	22	M	2 mo	N	N	N	Papilledema	28	11	1 y	Obesity

ACV thromb = acute cerebral venous thrombosis; Calcific SSS = calcifications of the brain scythe extended to SSS; CSF = cerebrospinal fluid; CT = computed tomography; ICP = intracranial pressure; MRI = magnetic resonance imaging; N = normal image/composition; RTS = right transverse sinus; SSS = superior sagittal sinus; tx = treatment.

anticoagulants in the remaining cases. Also, we used antiepileptic drugs for seizures in the case with acute cerebral venous thrombosis; antibiotics in the case with left mastoiditis and partial left transverse sinus thrombosis and symptomatic treatment [6,8]. One case with calcifications of the brain scythe extended to superior sagittal sinus and partial sinus stenosis (Fig. 2) initially had intracranial pressure (ICP) of 19 cmH_2O and after symptomatic treatment, acetazolamide and low-molecular-weight heparin, the ICP was 11 cmH_2O. It would be possible to place a venous sinus stent to prevent the extension of stenosis, but the follow-up period of the patient was only 3 months.

The hospitalization lasted for 3–5 weeks with clinical improvement after the therapy, including gradual improvement of the papillary edema. The ICP, which was determined before discharge, was 7–11 cmH_2O. The MRI controls after the treatment and before discharge showed the partial recanalization of these dural venous sinuses. The follow-up period was 1–12 months.

Discussion

The patients included in this study had headache, different stages of papilledema, elevated CSF pressure on lumbar puncture and normal CSF composition. The cerebral CT and MR images did not show a tumor, hydrocephalus, or other etiology, and the diagnosis remained pseudotumor cerebri. According to Foley's classic definition, to Dandy's diagnosis criteria modified by Wall and to Friedman and Jacobson's diagnosis criteria, one of the diagnostic elements is the nonidentification of a cause for the ICP increase [2,7,9–12], and idiopathic ICH is a diagnosis of exclusion [13].

The group of 12 patients (group A) had all the characteristics to be included in idiopathic ICH. The second group of nine patients (group B) also had some of the characteristics of idiopathic ICH, but the cerebral CT and MR images showed a cerebral venous sinus thrombosis or stenosis. Therefore, group B was diagnosed with ICH caused by intracranial vascular damage [14,15]. The comparison of the characteristics of the two patient groups revealed differences that enable a secure diagnosis, which is important because the treatments are different for the two types of ICH. The period from the first clinical signs until the complete clinical syndrome in group A was 6 weeks to 7 months, with mean 3 months. This period was only 5 days to 2 months for group B, with mean 3 weeks. The complete clinical syndrome implied an increase in ICP, and therefore

Figure 1. Native brain computed tomography: small lateral ventricles in idiopathic intracranial hypertension.

this increase in ICP was faster in the cases with a vascular etiology of ICH and slower for idiopathic ICH. Additionally the values of ICP were higher in the patients with idiopathic ICH (group A) over a longer period than in the patients from group B. The increase in ICP was up to 30 cmH_2O for group A with a mean pressure of 27 cmH_2O and only up to 26 cmH_2O for group B with mean 21.8 cmH_2O; the mean pressure is different between two groups and it is consistent with data from literature [2,7,12,15]. Based on all ICP values, the critical ICP values in ICH caused by intracranial vascular damage were approximately 21 cmH_2O, and the critical ICP values in idiopathic ICH were higher, up to 27 cm H_2O. This difference indicates that the compensatory mechanisms are not effective due to the faster increase in ICP and that decompensation occurs at a lower ICP value in the cases of ICH caused by intracranial vascular damage.

We can designate the ICH caused by intracranial venous vascular damage as vascular ICH. The imaging investigations using cerebral CT, cerebral MRI and angioMRI with the venous phase showed obvious differences between the patients with idiopathic ICH and the patients with vascular ICH. All patients with idiopathic ICH had normal brain CT, brain MRI and angio-MRI results or only small ventricles on CT [16,17] (Fig. 1). The same investigations showed brain edema and intracranial vascular damage, such as thrombosis, partial thrombosis, stenosis of the intracranial venous sinus, or cerebral venous thrombosis, in group B (Figs. 3–6). The presence of brain edema and venous thrombosis may explain the pathogenic mechanisms involved in vascular ICH. Venous sinus outflow obstruction in vascular ICH reduces the venous sinus flow and secondarily decreases the returning venous circulation from the brain, resulting in the appearance of a venous stasis and the slowing of brain sanguine circulation. Additionally, the thrombosis or stenosis of the venous sinuses diminishes the drainage of the CSF. The decrease in the venous flux and decrease in the absorption of the CSF cause the brain edema. The diminished drainage of the CSF causes the increased ventricular accumulation of CSF with high intraventricular pressure, and hydrocephalic brain edema can then occur. The evolution is progressive, and ICH occurs [18].

With respect to the etiology, idiopathic ICH consists of an ICH syndrome without a noticeable etiology; there are only associated factors or confounding conditions as metabolic and endocrine disorders, hypovitaminosis A, and medications [1,2,4,10,12].

In this study, which included three men and nine women diagnosed with idiopathic ICH, the men were young (age 18 years, 19 years, and 22 years) and obese, whereas six women displayed obesity and a 21-year-old woman who used oral tetracycline for acne as possible associate factors involved in the development of idiopathic ICH. Female sex predominance is evident, and no other factor seems to have an etiological relationship with ICH, which is consistent with the data from the literature. The treatment was only symptomatic.

Four men and five women, aged 23–61 years, were diagnosed with vascular ICH, and the anamnesis and imaging findings indicated the cause of the increased ICP. Two young women presented a clinical syndrome of ICH during the postnatal period and were diagnosed with partial thrombosis of the sagittal superior sinus; they displayed good results after the treatment and were followed up for

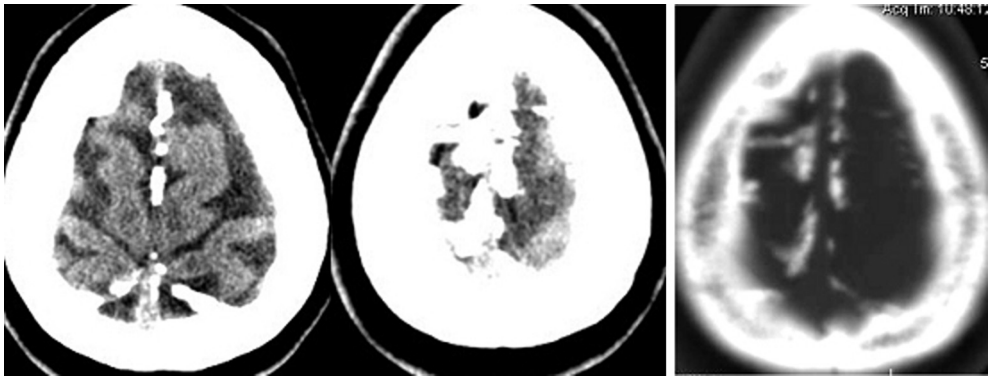

Figure 2. Native brain computed tomography: calcifications of the brain scythe extended to superior sagittal sinus with sinus stenosis in vascular intracranial hypertension.

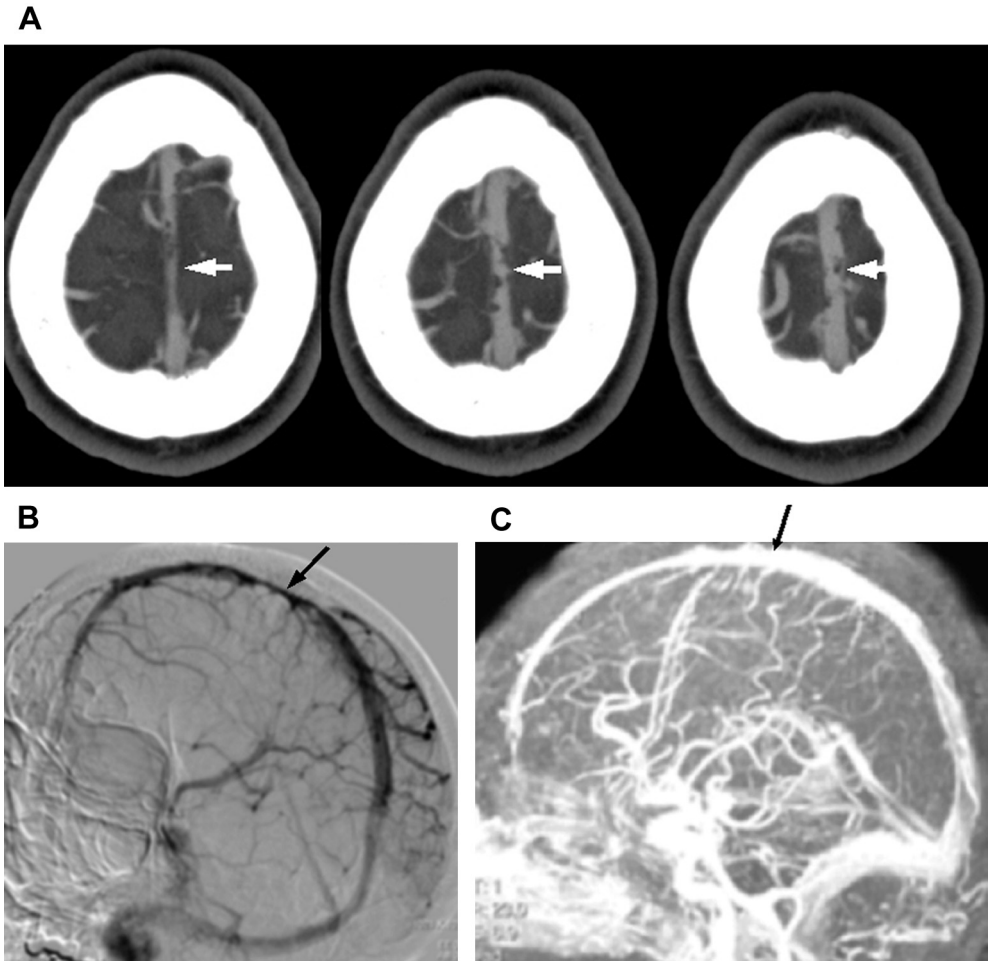

Figure 3. (A) Cerebral angiocomputed tomography: partial thrombosis of the superior sagittal sinus in vascular intracranial hypertension (ICH; arrow indicates partial thrombosis). (B) Cerebral Seldinger angiography: venous time with partial thrombosis of the superior sagittal sinus in vascular ICH (arrow indicates partial thrombosis). (C) Magnetic resonance venography with recanalization of the superior sagittal sinus after treatment in partial thrombosis of the superior sagittal sinus in vascular ICH (arrow).

6—12 months after their discharge. A 61-year-old man presented with chronic mastoiditis and developed a lateral venous sinus thrombosis on the same side (Fig. 6); he also had good results after antibiotic and anticoagulation therapy.

A 53-year-old man presented with acute ICH syndrome with 5 days of evolution before admission, and he was diagnosed with acute cerebral venous thrombosis with some characteristics of thrombophlebitis. He also presented with seizures, and the treatment immediately solved the syndrome. He was followed up for 9 months with good results. A 47-year-old man had progressive ICH syndrome for 10 weeks, and the imaging diagnosis showed calcifications of the brain scythe extending to the superior sagittal sinus, with sinus stenosis (Fig. 2); he only required symptomatic therapy. These calcifications obviously occurred over time, with some event triggering the ICH syndrome, which may have been the degree of venous sinus stenosis. The other patients had partial thrombosis of the superior sagittal sinus, and anticoagulation therapy provided good results, with recanalization of these dural venous sinuses on MRI control demonstrated before discharge (Fig. 3 B, C).

There were three cases with venous sinus stenosis and subacute ICH syndrome with significant improvement after symptomatic treatment and follow-up for 6 months. In venous sinus stenosis, there are discussions regarding whether the venous outflow obstruction is the etiology in some cases of ICH, and thus, this situation is included in vascular ICH. In contrast some authors allege that venous sinus stenosis is the result and not the cause of elevated ICP [19,20]. If the venous sinus stenosis is the cause of the vascular ICH, then the etiologic treatment is therapeutic stent placement in the venous sinuses [21,22]. This problem has been discussed, and the results of stent placement in the venous sinus are inconclusive [23,24]. Also, Karahalios et al suggested that increased intracranial venous pressure may be a universal mechanism in pseudotumor cerebri of different etiologies [25], but it seems that it is secondary to venous sinus stenosis or thrombosis and this is the mechanism in vascular ICH [26]. Idiopathic ICH has yet undiscovered mechanisms, which must ensure the intracranial circulatory autoregulation despite of the increased ICP.

The presentation and analysis of these cases allowed the differentiation between the vascular type of ICH and idiopathic ICH. The name hyperemic ICH has been suggested

Figure 4. Angio-magnetic resonance venous time: stenosis of the left transverse sinus in vascular intracranial hypertension (arrow indicates the stenosis).

[13], but the designation of vascular ICH covers the etiology and pathogenesis better and more completely.

Although the symptoms may be similar in these two diseases, the imaging findings, therapy and evolution are different; therefore, the etiology and pathogenesis are different. Vascular ICH has a known etiology, such as cerebral vascular illness whereas idiopathic ICH has no known

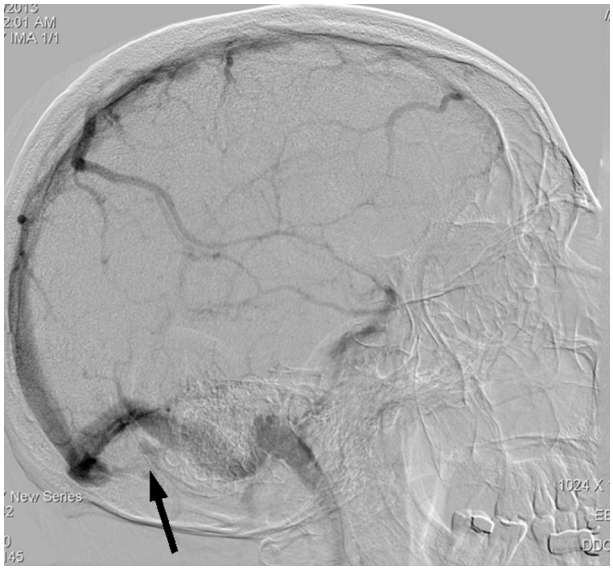

Figure 5. Cerebral Seldinger angiography: venous time with stenosis of the left transverse sinus in vascular intracranial hypertension (arrow indicates the stenosis).

Figure 6. Angio-magnetic resonance venous time: thrombosis of the left transverse sinus in vascular intracranial hypertension (arrow indicates thrombosis).

etiology or has various nonspecific factors associated factors (e.g., metabolic and endocrine disorders, hypovitaminosis A, and medications). The imaging diagnosis for vascular ICH showed a cerebral venous sinus thrombosis or stenosis or a cerebral venous thrombosis and either normal images or small ventricles in idiopathic ICH. The imaging diagnosis of idiopathic ICH excludes other diseases with similar symptoms.

Vascular ICH involves vasogenic brain edema with papillary edema, whereas idiopathic ICH commonly involves papillary edema and diminished visual acuity in some cases. There is also brain edema in idiopathic ICH, but the brain edema appears to be balanced by the intraventricular pressure [18].

The increase in ICP is faster in vascular ICH compared with the very slow ICP increase in idiopathic ICH, as demonstrated by the longer period until the complete clinical syndrome has developed. In addition, the ICP values are higher in idiopathic ICH. Therefore, the critical ICP values are lower in vascular ICH until the decompensation of ICH.

The treatment is symptomatic, as well as etiologic and pathogenic in vascular ICH but only symptomatic in idiopathic ICH (including a lumboperitoneal shunt or decompression of the optic nerve).

Vascular ICH may potentially include other syndromes, such as hypertensive encephalopathy, but this classification requires further study. In hypertensive encephalopathy, there is a dilatation of brain arteries with increased arterial inflow, increased vascular permeability, and disruption of the brain—blood barrier; brain edema occurs as a result [27,28].

In conclusion, ICH caused by intracranial vascular damage can be called vascular ICH. Our results and the data from the literature support that, although it has clinical similarities to idiopathic ICH, there are important

differences: vascular ICH has a known etiology, such as cerebral vascular illness, and the increase in ICP is faster in vascular ICH, but the critical ICP values are lower compared with idiopathic ICH. The therapy is etiologic, pathogenic and symptomatic in vascular ICH, but is only symptomatic in idiopathic ICH.

References

[1] Pearce JM. From pseudotumour cerebri to idiopathic intracranial hypertension. Pract Neurol 2009;9:353—6.
[2] Johnston I. The historical development of the pseudotumor concept. Neurosurg Focus 2001;11:1—9.
[3] Degnan AJ, Levy LM. Pseudotumor cerebri: brief review of clinical syndrome and imaging findings. AJNR Am J Neuroradiol 2011;32:1986—93.
[4] Farb RI, Vanek I, Scott JN, Mikulis DJ, Willinsky RA, Tomlinson G, et al. Idiopathic intracranial hypertension: the prevalence and morphology of sinovenous stenosis. Neurology 2003;60:1418—24.
[5] Brazis PW. Pseudotumor cerebri. Curr Neurol Neurosci Rep 2004;4:111—6.
[6] Sylaja PN, Ahsan Moosa NV, Radhakrishnan K, Sankara Sarma P, Pradeep Kumar S. Differential diagnosis of patients with intracranial sinus venous thrombosis-related isolated intracranial hypertension from those with idiopathic intracranial hypertension. J Neurol Sci 2003;215:9—12.
[7] Friedman DI, Jacobson DM. Diagnostic criteria for idiopathic intracranial hypertension. Neurology 2002;59:1492—5.
[8] Einhäupl K, Stam J, Bousser MG, De Bruijn SF, Ferro JM, Martinelli I, et al. EFNS guideline on the treatment of cerebral venous and sinus thrombosis in adult patients. Eur J Neurol 2010;17:1229—35.
[9] Wall M. Idiopathic intracranial hypertension. Neurol Clin 2010; 28:593—617.
[10] Digre KB, Nakamoto BK, Warner JE, Langeberg WJ, Baggaley SK, Katz BJ. A comparison of idiopathic intracranial hypertension with and without papilledema. Headache 2009; 49:185—93.
[11] Biousse V, Bruce BB, Newman NJ. Update on the pathophysiology and management of idiopathic intracranial hypertension. J Neurol Neurosurg Psychiatry 2012;83:488—94.
[12] Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 2013;81:1159—65.
[13] Bateman GA. Vascular hydraulics associated with idiopathic and secondary intracranial hypertension. AJNR Am J Neuroradiol 2002;23:1180—6.
[14] Gross CE, Tranmer BI, Adey G, Kohut J. Increased cerebral blood flow in idiopathic pseudotumor cerebri. Neurol Res 1990;12:226—30.
[15] Leker RR, Steiner I. Features of dural sinus thrombosis simulating pseudotumor cerebri. Eur J Neurol 1999;6:601—4.
[16] Suzuki H, Takanashi J, Kobayashi K, Nagasawa K, Tashima K, Kohno Y. MR imaging of idiopathic intracranial hypertension. Am J Neuroradiol 2001;22:196—9.
[17] Hoffmann J, Huppertz HJ, Schmidt C, Kunte H, Harms L, Klingebiel R, et al. Morphometric and volumetric MRI changes in idiopathic intracranial hypertension. Cephalalgia 2013;33: 1075—84.
[18] Iencean SM. Simultaneous hypersecretion of CSF and of brain interstitial fluid causes idiopathic intracranial hypertension. Med Hypotheses 2003;61:529—32.
[19] Lee SW, Gates P, Morris P, Whan A, Riddington L. Idiopathic intracranial hypertension; immediate resolution of venous sinus "obstruction" after reducing cerebrospinal fluid pressure to $<10 cm H_2O$. J Clin Neurosci 2009;16:1690—2.
[20] Stienen A, Weinzierl M, Ludolph A, Tibussek D, Häusler M. Obstruction of cerebral venous sinus secondary to idiopathic intracranial hypertension. Eur J Neurol 2008;15:1416—8.
[21] Kumpe DA, Bennett JL, Seinfeld J, Pelak VS, Chawla A, Tierney M. Dural sinus stent placement for idiopathic intracranial hypertension. J Neurosurg 2012;116:538—48.
[22] Bateman G. Stenoses in idiopathic intracranial hypertension: to stent or not to stent? ANJR Am J Neuroradiol 2008;29:215.
[23] Rohr A, Dörner L, Stingele R, Buhl R, Alfke K, Jansen O. Reversibility of venous sinus obstruction in idiopathic intracranial hypertension. AJNR Am J Neuroradiol 2007;28:656—9.
[24] Lobo S, Ferro JM, Barinagarrementeria F, Bousser MG, Canhão P, Stam J, et al. Shunting in acute cerebral venous thrombosis: a systematic review. Cerebrovasc Dis 2013;37: 38—42.
[25] Karahalios DG, Rekate HL, Khayata MH, Apostolides PJ. Elevated intracranial venous pressure as a universal mechanism in pseudotumor cerebri of varying etiologies. Neurology 1996;46:198—202.
[26] Kim AW, Trobe JD. Syndrome simulating pseudotumor cerebri caused by partial transverse venous sinus obstruction in metastatic prostate cancer. Am J Ophthalmol 2000;129: 254—6.
[27] Kanazawa M, Sanpei K, Kasuga K. Recurrent hypertensive brainstem encephalopathy. J Neurol Neurosurg Psychiatry 2005;76:888—90.
[28] Lee S, Cho BK, Kim H. Hypertensive encephalopathy with reversible brainstem edema. J Korean Neurosurg Soc 2013;5: 139—41.